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Finley, Anna; Angus, D; van Reekum, Carien; Davidson, Richard; Schaefer, Stacey

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**Cross-Sectional Associations Between Theta-Beta Ratios and Individual Peak  
Alpha Frequency Across Adulthood**

Anna J. Finley, Douglas J. Angus, Carien M. van Reekum, Richard Davidson, Stacey  
M. Schaefer

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Correspondence concerning this article should be addressed to Anna J. Finley, University of Wisconsin, Madison. Email: ajfinley2@wisc.edu.

### Abstract

Prior research has found the ratio of fronto-central theta (4–7 Hz) and beta oscillations (13–30 Hz), known as the theta-beta ratio, to be negatively correlated with attentional control, reinforcement learning, executive function, and age. While theta-beta ratios have been found to decrease with age in adolescents and undergraduate samples, theta has been found to increase with age in older adults. Moreover, age-related decreases in individual peak alpha frequency may be artifactually inflating theta with age. Collectively, these factors lead to an incomplete understanding of how theta-beta ratio varies across the lifespan, particularly in older adults. We conducted a preregistered analysis of data from the Midlife in the US (MIDUS) study examining the cross-sectional associations between resting theta-beta ratio, age, and individual peak alpha frequency ( $n = 264$ ; age 36–84,  $M = 55.7$ ,  $SD = 11.0$ ). Age was negatively correlated with theta-beta ratios and individual peak alpha frequencies. Although we also observed a significant negative correlation between theta and individual peak alpha frequencies, the relationship between theta and age was only apparent in exploratory analyses controlling for individual peak alpha frequency and beta. Notably, the correlation between theta-beta ratios and age remained after controlling for individual peak alpha frequencies. Our results replicate previous observations that theta-beta ratios and individual peak alpha frequencies are cross-sectionally associated with age, and show that age-related decreases in theta-beta ratios are not due to age-related decreases in individual peak alpha frequencies.

Keywords: Theta; Beta; Theta-Beta Ratio; Individual Peak Alpha Frequency; Aging

## **1 Introduction**

The use of spontaneous resting electroencephalographic (EEG) activity as an objective measure of individual differences in psychological functioning has a long history (Davidson, 1984; Klimesch, 1999; Knyazev, 2007; Schutter & Knyazev, 2012). Resting EEG has typically been divided into bands based on spectral power, from the slowest frequencies in delta (0-4 Hz), through theta (4-7 Hz), alpha (7-13 Hz), beta (13-30 Hz), to the fastest frequencies in gamma (30+ Hz). A persistent but growing focus is on the use of these resting EEG measures as biomarkers for optimal and sub-optimal executive function, particularly in the context of identifying healthy vs. unhealthy executive function development and decline with aging (Arns et al., 2013; Babiloni et al., 2006). Extant evidence suggests that individual and intra-individual differences in executive function broadly, and attentional control specifically, are associated with the ratio between fronto-central theta and beta oscillations (Angelidis et al., 2016).

### **1.1 Theta-Beta Ratios**

The ratio between fronto-central theta and beta oscillations has emerged as a marker of executive [dys]function associated with Attention-Deficit Hyperactive Disorder (ADHD; Arns et al., 2013) where higher ratios – indicating relatively greater fronto-central theta than fronto-central beta - are characteristic of ADHD. In non-clinical populations, theta-beta ratio has been found to be strongly negatively correlated with self-report and behavioral measures of executive function, while theta and beta alone are not (Perone et al., 2018), and smaller theta-beta ratios are indicative of better cognitive control, executive control, and increased vigilance (Angelidis et al., 2016, 2018; Putman et al., 2010, 2014; van Son, Schalbroeck, et al., 2018). Several studies have shown that beyond being a marker of attentional control, theta-beta ratios are negatively correlated with response-inhibition by threatening stimuli (Putman et al., 2010) and are positively correlated with attentional capture by mildly

threatening stimuli relative to highly threatening stimuli (van Son, Angelidis, et al., 2018). Theta-beta ratios have also been found to negatively correlate with advantageous decision making in reinforcement learning paradigms (Massar et al., 2014; Schutter & Van Honk, 2005). Other studies have reported that theta-beta ratios predict risk taking behavior, and that theta and beta alone do not (Massar et al., 2012). Recent studies have also shown that theta-beta ratios increase during mind-wandering (van Son, De Blasio, et al., 2019; van Son, de Rover, et al., 2019). Together, these studies suggest that theta-beta ratios are related to attentional control broadly, as well as to more specific emotional and rewarding contexts, such that lower theta-beta ratios are reflective of more control or focus.

Theta-beta ratios are argued to reflect the reciprocal regulation of bottom-up subcortical processes by top-down cortical processes (Knyazev, 2007; Schutter & Knyazev, 2012). Although much of the research supporting this subcortical-cortical model of theta-beta ratios and executive function has been indirect, a recent study has provided direct support (van Son, de Rover, et al., 2019). In a landmark study, van Son et al., (2019) showed that not only are theta-beta ratios lower when participants exert attentional control compared to when they engage in mind-wandering, but that these changes are associated with decreased functional connectivity between dorsolateral prefrontal cortex (DLPFC) and the dorsal anterior cingulate cortex (ACC) – regions which have been associated with executive function in multiple domains (Seeley et al., 2007).

Given age-related decline in executive function (Buckner, 2004; Lustig & Jantz, 2015) and in cortical integrity (Fjell et al., 2017; Madden et al., 2009, 2012), one would assume a straight-forward relationship between theta-beta ratio and age. The mapping between age and the theta-beta ratio is more complicated, however. First, theta has been observed to *increase* with age, potentially through a migration of alpha activity to the upper frequencies of theta, discussed in more detail below (Klimesch, 1999). Second, resting theta

recorded from the same scalp locations used in theta-beta ratio research has been positively correlated with cognitive function in older adults (73 adults ages 56-70, Finnigan & Robertson, 2011; 53 adults ages 18-89, Vlahou et al., 2014). Third, in child and young adult samples, theta-beta ratios have been reported to be negatively correlated with age (41 young adults ages 18-31 years, Angelidis et al., 2016; 41 children ages 8-12 years, Clarke et al., 2001; 101 children ages 7-16 years, Ogrim et al., 2012; 162 children ages 3-9 years, Perone et al., 2018; 28 young adults ages 19-28 years, Putman et al., 2010, but see Putman et al., 2014 for a non-replication in 77 young adults with a mean age of 19.9 years), and with cognitive function (Angelidis et al., 2016, 2018; Putman et al., 2010, 2014; 128 young adults with a mean age of 22.3 years, Schutte et al., 2017).

## **1.2 Individual Peak Alpha Frequencies**

Individual peak alpha frequency is the frequency at which power in the alpha band (7-13 Hz) peaks, has been found to be negatively correlated with age in adulthood (Clark et al., 2004; Klimesch, 1997), and is reduced in individuals with Alzheimer's disease (Klimesch, 1997). Research has associated higher individual peak alpha frequency across adulthood with better working memory, better reading comprehension, and a larger general intelligence factor (Angelakis et al., 2004; Clark et al., 2004; Grandy et al., 2013; Klimesch, 1997), suggesting it is an indicator of cognitive capacity or preparedness.

Alpha power has been associated with reductions in blood flow across wide areas of the frontal and parietal cortex (Jensen & Mazaheri, 2010; Laufs et al., 2003). During working memory tasks, greater local alpha power during a trial was predictive of better memory performance (Jensen & Mazaheri, 2010), and decreases in BOLD activation, particularly in areas of the default mode network, are also related to working memory performance (Anticevic et al., 2010; Daselaar et al., 2004). Together, these findings suggest that alpha

power indexes the ability to inhibit task-irrelevant regions while performing cognitive tasks (Jensen & Mazaheri, 2010).

With regards to theta-beta ratios, because the peak alpha power “slows”, or shifts into lower frequencies with age, some of the EEG power associated with the alpha band (commonly defined as 7-13 Hz) may be mistakenly attributed to power in the theta band (4-7 Hz) in older adults, driving increases in theta with age and therefore changes in theta-beta ratios. While some work suggests that relative theta in older adults may be positively correlated with measures of memory, attention, and executive functioning, the potential role of alpha leaking into the theta band, as indexed by individual peak alpha frequency, remains unclear (Finnigan & Robertson, 2011). Therefore, understanding how resting theta, beta, theta-beta ratios, and individual peak alpha frequencies are cross-sectionally interrelated across the adult age-span is key to beginning to understand how these EEG metrics relate to healthy aging.

### **1.3 The Present Study**

As reviewed above, theta-beta ratios have been found to be negatively associated with age, such that larger ratios – indicating relatively greater theta power than beta power - are observed in samples of younger participants compared to older participants. However, child and young adult samples with restricted age ranges (e.g., children 3-9 years old in Perone et al., 2018; young adults 19-28 years old in Putman et al., 2010) currently predominate the studies of age-related differences in theta-beta ratios. In the present study, we extend the research on theta-beta ratios and age to include older adults by examining whether the negative association between age and theta-beta ratios is observed in a large sample featuring a wide adult age range (from 36 to 84 years). Additionally, we examine to what extent any associations between theta-beta ratios and age are accounted for by age-related differences in individual peak alpha frequency.

We conducted a preregistered secondary analysis of data from the Midlife in the US Study's Neuroscience Project (MIDUS 2, 2004-2009; <http://midus.wisc.edu/>), examining whether the correlation between theta-beta ratios and age is due to variation in associations between age and theta, age and beta, or age-related decreases in individual peak alpha frequencies. Based on previous studies, we developed and tested two pre-registered hypotheses. First, we tested whether the negative correlation between theta-beta ratios and age is replicated in a large sample of older adults ranging in age from 36 to 84 years old, predicting that greater age will be correlated with lower theta-beta ratios. Second, we used the RestingIAF package (<https://github.com/corcorana/restingIAF>; Corcoran et al., 2018) to test whether there would be a pattern of alpha “slowing” with age, predicting that older age would be associated with lower individual peak alpha frequencies. We also explored the extent to which theta and beta are unique predictors of age at time of recording, and the extent to which theta-beta ratios and age are linearly related. Lastly, we examined whether the correlation between theta-beta ratios and age was preserved when statistically adjusting for individual differences in individual peak alpha frequencies, and examined if the relationships held controlling for gender and race<sup>1</sup>. All analyses and hypotheses were preregistered prior to the extraction of new EEG frequency metrics and their statistical analysis at <https://osf.io/n57au>. Additionally, the new EEG reprocessing pipeline was registered here <https://osf.io/wfkmn>.

## 2. Method

### 2.1 Participants

The present study used data collected during the second wave of Midlife in the US (MIDUS) in the Neuroscience Project (2004-2009), consisting of 331 participants from the

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<sup>1</sup> We did not explicitly register the exploratory analyses between theta-beta ratios and individual peak alpha frequencies, nor did we explicitly register controlling for participant race.



main MIDUS cohort. These respondents included three distinct subsamples: the Main Longitudinal (n = 135), Twin (n = 88) and Milwaukee (n = 108) subsamples (see <http://midus.wisc.edu/midus2/project5/> for additional details about sampling strategies within these subsamples). The Main Longitudinal and Twin subsamples contained individuals who participated in the initial wave of MIDUS data collection approximately 10 years prior. The Milwaukee subsample contained individuals who participated in the baseline MIDUS Milwaukee study, initiated in 2005. Demographic information is presented in Table 1, and sensitivity power analyses are reported in the supplemental materials.

All data collection procedures were approved by the UW-Madison Institutional Review Board, and informed consent was obtained for all participants. Participants with unusable resting spectral power EEG data (n = 15, 3.6%) and without identifiable peak alpha frequencies (n = 55, 16.7%) were excluded from analyses, yielding a final sample of n = 264 participants.

Table 1. Sample demographics

	Sufficient EEG data (n = 264)	Insufficient definable alpha peaks (n = 55)	Insufficient epochs for spectral power (n = 12)
Age in Years	55.7 (11.0)	53.1 (11.4)	58.8 (11.7)
Gender	120 male, 144 female	21 male, 34 female	7 male, 5 female
Race/Ethnicity	165 White, 89 Black, 4 Hispanic/Black, 1 Hispanic/White, 2 Asian, 3 Other	41 White, 11 Black, 2 Native American, 1 Other	6 White, 6 Black
Handedness	248 right, 16 left	50 right, 5 left	10 right, 2 left
MIDUS Subsample	105 Main, 66 Twin, 93 Milwaukee	25 Main, 19 Twin, 11 Milwaukee	5 Main, 3 Twin, 4 Milwaukee

## 2.2 Materials

### 2.2.1 Demographics

Demographic variables are publicly available via Colectica (<http://midus.colectica.org/>) and the Inter-university Consortium for Political and Social Research (ICPSR; <https://www.icpsr.umich.edu/web/ICPSR/series/203>). From the MIDUS 2 Neuroscience Project dataset, we used age at time of EEG data collection, gender, race (dichotomized as White/non-White for analyses) and Family ID. Family ID was used to

account for genetic dependencies in follow-up analyses in the supplemental materials. See Table 1 for a breakdown of demographics.

## **2.3 Procedure**

### **2.3.1 EEG Recording**

EEG data were collected using a 128 channel geodesic net of Ag/AgCl electrodes encased in saline dampened sponges (Electrical Geodesics, Inc [EGI], Eugene, OR) with impedances reduced to less than 100 K $\Omega$  whilst ensuring that electrolyte “bridges” (see Greischar et al., 2004) had not formed. After the net was placed, participants were escorted into a soundproof booth where they were seated in front of a computer screen. A computer located outside the booth recorded the data. Signals were amplified and sampled at 500 Hz with an online bandpass filter from 0.1-100 Hz at 16-bit precision using an online vertex (Cz) reference. The participant was instructed to rest for six 1-minute periods. During three of the 1-minute periods they were asked to keep their eyes open; for the remaining three 1-minute periods they were asked to keep their eyes closed. The order of the eyes open/eyes closed was pseudorandomized, with two fixed orders counterbalanced across participants. Participants then completed an emotional picture viewing task (data not presented here), followed by another baseline resting recording for six 1-minute periods. Prior data processing was restricted to alpha asymmetry variables from the first baseline recording, collapsed across the entire 6-minute period (e.g., Hostinar et al., 2017). The current analyses focus on metrics extracted from the first resting recording.

## **2.4 Data Reduction**

### **2.4.2 EEG preprocessing**

Offline the EEG data was filtered (60 Hz notch, 0.5 Hz high-pass), bad channels identified and removed, and bad sections of data identified and removed. EEGLab6 was then used to conduct a PCA/ICA to identify 20 components, which were screened to remove

obvious blink, eye movement, and other artifacts. Bad channels were replaced using a spherical spline interpolation. Data from the eyes open and eyes closed conditions were collapsed. These are the original preprocessing steps from the initial alpha asymmetry pipeline that were preserved in the reprocessing pipeline.

### **2.4.3 EEG reprocessing: Spectral power for theta-beta ratio**

All data reprocessing was completed using EEGLab 2019.1 scripts implemented in MATLAB 2019b. Data was re-referenced to the average reference and Cz was imputed. Continuous resting data was epoched into 2 second segments with 50% overlap, and bad segments were rejected if there was a voltage deviation on any channel of  $\pm 100\mu\text{V}$ . Participants with more than 50% of the total number of epochs rejected were excluded from analyses in a listwise fashion ( $n = 12$ ). EEG spectral power at each predefined spectral band (theta: 4-7 Hz; alpha: 7-13 Hz; beta: 13-30 Hz) was extracted using a 2 second Hamming window padded by a factor of 2 with 50% overlap. Spectral power was extracted individually for each channel, then averaged over sensors to map onto the standard 10-20 channels as shown in the reprocessing pipeline registration (<https://osf.io/wfkmn>). Theta and beta values were calculated from the initial baseline recording as the average composite of the F3/Fz/F4 analog channels and were transformed to a theta-beta ratio by dividing the former by the latter and subsequently log-normalized.

### **2.4.4 EEG reprocessing: Individual peak alpha frequency**

Individual peak alpha frequency from the initial baseline recording was extracted using the RestingIAF package (<https://github.com/corcorana/restingIAF>; Corcoran et al., 2018), using adjustments to the parameters based on our sample of older adults as recommended by Corcoran et al (2018). We used a 2 second Hamming window with 50% overlap,  $F_w = 11$  (frequency span of  $\sim 2.69$  Hz);  $k = 4$ ;  $W_\alpha = [6, 14 \text{ Hz}]$ ;  $f\text{Range} = [1, 40 \text{ Hz}]$ ,  $\text{mpow} = 0.6$ ,  $p\text{Diff} = 0.20$ ,  $c\text{Min} = 3$ . Those individuals who did not exhibit a clear peak

value in 50% of the sensors used for the composites or 50% of the overall scalp were excluded from analyses in a listwise fashion ( $n = 55$ ). Estimates were extracted individually for each channel, averaged over sensors to map onto the standard 10-20 channels as shown in the reprocessing pipeline registration (<https://osf.io/wfkmm>), as well as an overall average peak alpha power computed across all channels. Individual peak alpha frequency for the current study was quantified as the average composite of the F3/Fz/F4 analog channels.

### 3. Results

#### 3.1 Confirmatory Analyses

Our first hypothesis was initially tested using Pearson's Correlations between log-normalized theta-beta ratios and age. As shown in Table 2 above the diagonal and in Figure 1, resting theta-beta ratios were negatively correlated with age ( $r = -0.22$ , 95% CI  $[-0.33, -0.10]$ ,  $p < 0.001$ ), such that the ratio of slow-wave to fast-wave activity was lower for older participants. Consistent with a prior unpublished analysis of this data set and previous independent studies in adults (Clark et al., 2004; Klimesch, 1999), we also observed a significant negative correlation between individual peak alpha frequency and age ( $r = -0.19$ , 95% CI  $[-0.30, -0.07]$ ,  $p = 0.008$ ), such that peak alpha frequencies were lower in older participants (Table 2 above the diagonal, Figure 1). Generalized Estimating Equations (GEE) confirmed these relationships held when adjusting for genetic dependencies between twin participants ( $n = 66$ ; Supplemental Table 1 for details of the GEE analyses). We also examined the partial correlations controlling for gender, and still observed a significant negative correlation between age and theta-beta ratio ( $r = -0.21$ , 95% CI  $[-0.33, -0.10]$ ,  $p < 0.001$ ), as well as between age and individual peak alpha frequency ( $r = -0.19$ , 95% CI  $[-0.31, -0.07]$ ,  $p < 0.001$ ).

Table 2. Correlations between age and EEG metrics.

	Age	Theta	Beta	Theta-Beta Ratio	Individual Peak Alpha Frequency
Age	--	0.01 [-0.11, 0.13] $p = .876$	0.21 [0.09, 0.33] $p = .002$	-0.22 [-0.33, -0.10] $p < .001$	-0.19 [-0.30, -0.07] $p = .008$
Theta	-0.07 [-0.19, -0.05] $p = .257$	--	0.42 [0.31, 0.51] $p < .001$	0.51 [0.41, 0.59] $p < .001$	-0.40 [-0.50, -0.29] $p < .001$
Beta	0.20 [0.08, 0.31] $p = .001$	0.42 [0.32, 0.51] $p < .001$	--	-0.26 [-0.37, -0.15] $p < .001$	-0.10 [-0.22, 0.02] $p = .223$
Theta-Beta Ratio	-0.34 [-0.44, -0.23] $p < .001$	0.41 [0.30, 0.50] $p < .001$	-0.34 [-0.44, -0.34] $p < .001$	--	-0.43 [-0.52, -0.32] $p < .001$

Note: Shaded values above the diagonal represent Pearson correlations and 95%

confidence intervals. Unshaded values below the diagonal represent partial correlations and 95% confidence intervals controlling for individual peak alpha frequency.

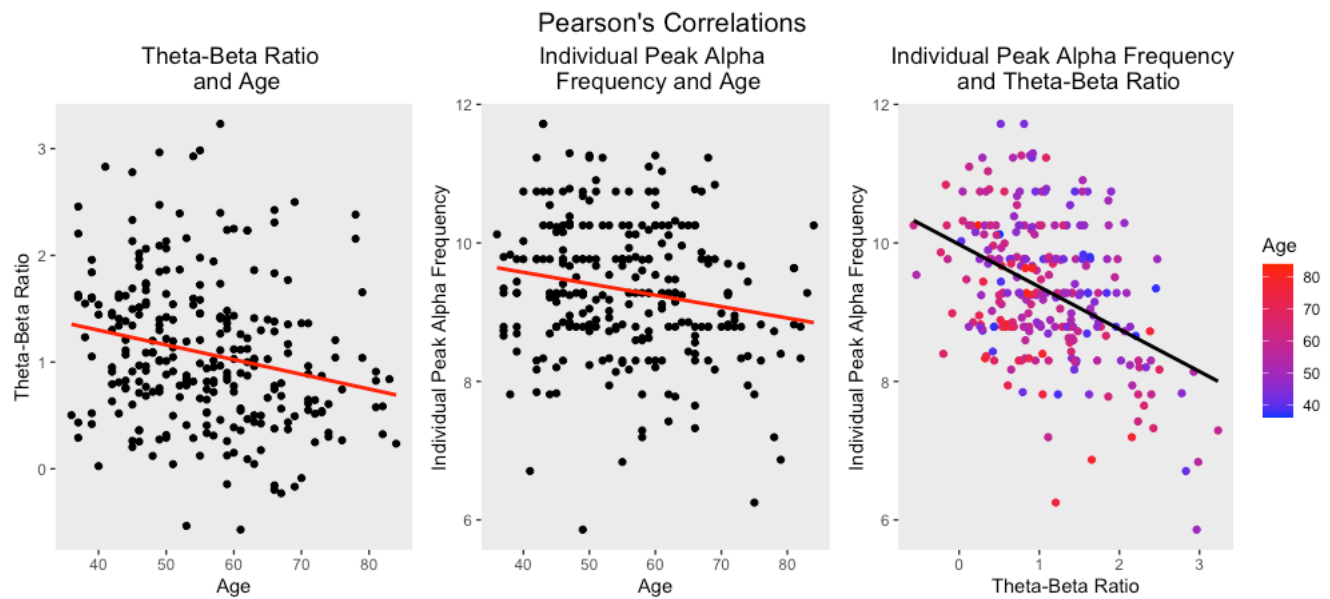


Figure 1. Pearson's correlation scatterplots between theta-beta ratio, individual peak alpha frequency, and age.

### 3.2 Exploratory Analyses

Visual inspection of the association between theta-beta ratios and age, and individual peak alpha frequencies suggest that within this sample, these associations are linear. As such, we conducted linear exploratory analyses as described in our preregistration (<https://osf.io/n57au>). Exploratory analyses indicated that the association between theta-beta ratios and age remained even when statistically adjusting for the age-related slowing in individual peak alpha frequency (Table 2 unshaded below the diagonal, Figure 2), suggesting that age related changes in theta-beta ratio are independent of shifts in individual peak alpha frequency.

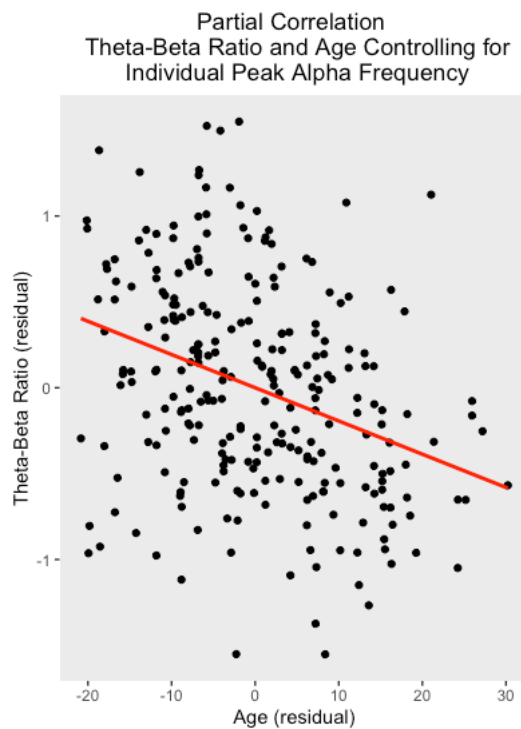


Figure 2. Partial correlation scatterplot between theta-beta ratio and age, controlling for individual peak alpha frequency.

Our primary preregistered exploratory aim was to determine the extent to which theta and beta have unique associations with age, which we examined using general linear models predicting age from theta and beta, with individual alpha variables added at the second block. As shown in Table 3, when included alone only frontal beta power was associated with age,  $b = 15.49$ ,  $p < .001$ . However, when individual peak alpha frequency was also included, all three measures were significantly associated with age. This suggests the relationship between theta power and age is obscured by the relationship between individual peak alpha frequency slowing and age, pointing to the importance of controlling for individual peak alpha frequency when considering power in the theta band in aging.



Table 3. Step-wise multiple regression predicting age.

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>p</i>	Adj. <i>R</i> <sup>2</sup>
Block 1					0.046
Theta	-0.83	0.57	1.46	.146	
Beta	15.49	4.05	3.83	< .001	
Block 2					0.090
Theta	-1.72	0.61	2.83	0.005	
Beta	16.70	3.97	4.21	< .001	
Individual					
Peak Alpha	-2.67	0.72	3.70	< .001	
Frequency					

Finally, we also examined the partial correlations controlling for gender and race (dichotomized as White/non-White), and still observed a significant negative correlation between age and theta-beta ratio ( $r = -0.21$ , 95% CI [-0.32, -0.09],  $p < 0.001$ ), as well as between age and individual peak alpha frequency ( $r = -0.20$ , 95% CI [-0.32, -0.08],  $p < 0.001$ ).

#### 4. Discussion

In the current preregistered study, we aimed to replicate and extend previous observations that theta-beta ratios and individual peak alpha frequency are associated with age in a large sample of 264 adults featuring a wide age range (36-84 years). Consistent with predictions and previous studies, we found that both theta-beta ratios and individual peak alpha frequencies were negatively correlated with age. Exploratory analyses indicated that the association between theta-beta ratios and age remained when controlling for individual peak

alpha frequencies, demonstrating that age-related decreases in theta-beta ratios are not entirely due to age-related decreases in individual peak alpha frequencies.

#### **4.1 Understanding how TBRs and IPAFs change over the lifespan**

Notably, the association between theta-beta ratios and age remained when accounting for age related difference in individual peak alpha frequencies, and the relationship between theta power and age is only apparent when controlling for both beta power and individual peak alpha frequency. This suggests that both shifts in individual peak alpha frequencies and theta-beta ratios are independently related to aging, and that once variance between age and individual peak alpha frequency is accounted for both a decrease in theta and increase in beta drive the relationship between theta-beta ratios and aging. The current study is limited to examining the cross-sectional relationships between frontal measures of resting EEG with age in adulthood across a span of nearly 50 years. Additional longitudinal research is needed to examine the developmental trajectory of these metrics within individuals.

#### **4.2 Implications for theta-beta ratio and individual peak alpha frequency as markers of executive function and healthy aging**

Although our findings supported our hypotheses, they complicate the interpretation of theta-beta ratio as a marker of executive function. In adolescents and young adults, higher theta-beta ratios are associated with more executive dysfunction and related to ADHD (Arns et al., 2013), and lower theta-beta ratios are associated with better attentional control (Perone et al., 2018). Considering older age-related decline in executive function (Buckner, 2004; Lustig & Jantz, 2015), theta-beta ratios may exhibit a curvilinear relationship with executive functioning, such that better executive functioning is related to a moderate level of theta-beta ratio. Additionally, it may be that adolescence and younger adults are more prone to disruptions related to elevated theta-beta ratios and older adults are more prone to reductions in theta-beta ratios potentially driven by normative aging processes. The moderate level of

theta-beta ratio may reflect an optimal balance in the bidirectional regulation of bottom-up subcortical processes by top-down cortical processes that theta-beta ratio is putatively suggested to index (Knyazev, 2007; Schutter & Knyazev, 2012). Additional longitudinal work across the human lifespan is needed to clarify the developmental trajectory of theta-beta ratios, and confirm it tracks with changes in cortical and subcortical bidirectional regulation.

Given the unique relationships between individual peak alpha frequency and theta-beta ratio with age, as well as existing research linking individual peak alpha frequency with memory-related aspects of executive functioning (i.e., Clark et al., 2004) and theta-beta ratio with attention-related aspects of executive functioning (Angelidis et al., 2016), these markers appear to be promising, dissociable measures of healthy aging. Further research is needed to confirm the unique predictive power of individual peak alpha frequency and theta-beta ratio with memory, executive functioning, and measures of healthy and pathological aging.

## **5. Conclusion**

Overall, we found that both theta-beta ratios and individual peak alpha frequencies were cross-sectionally negatively associated with age, and that age-related decreases in theta-beta ratios are not due to age-related decreases in individual peak alpha frequencies. This suggests that changes in both theta-beta ratios and individual peak alpha frequencies may index differential components of healthy aging. Future research should explicitly examine multiple facets of executive function (including working memory, attention control, and response inhibition) to determine how theta-beta ratios and individual peak alpha frequencies at rest relate to cognitive functioning in older adulthood, and if these measures are suitable as biomarkers for healthy and pathological aging. Additionally, we are limited by the cross-sectional nature of the study from determining if these cross-sectional relationships between age and resting EEG metrics reflect an underlying developmental trajectory in aging. Future

longitudinal research is needed to trace the developmental trajectory of theta-beta ratios and individual peak alpha across the lifespan.

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## **Supplemental Materials**

### **S1. Sensitivity power analysis**

We used G\*Power 3.1 (Faul et al., 2009) to conduct sensitivity power analysis prior to data reprocessing for a sample of 300 participants as an estimate for the final usable sample size after applying our criteria for usable EEG data (i.e., 50% of epochs retained for the spectral power density, 50% of channels resulting in definable alpha peaks). This analysis indicated that we would have 95% power to detect a Pearson's correlation of .20, and 95% power to detect a small to medium sized effect in regression analyses ( $f^2 = .06$ ).

### **S2. Generalized estimating equations analyses**

To ensure our findings were not spuriously due to genetic interdependencies due to the inclusion of 66 individuals from the MIDUS Twin subsample, we conducted parallel analyses controlling for families using general estimating equations with the R gee: Generalized Estimation Equation Solver package (Carey et al., 2019; <https://cran.r-project.org/web/packages/gee/index.html>). As shown in Table S1, age was still significantly negatively related to TBR and IPAF, and IPAF and TBR were also still significantly negatively related.

Table S1. General estimating equations controlling for genetic dependencies across twins.

	<i>b</i>	<i>Naïve SE</i>	<i>Naïve z</i>	<i>Naïve p</i>
Age:				
Theta-Beta Ratio	-5.9	1.02	5.8	< .001
Individual Peak				
Alpha Frequency	-3.8	0.71	5.4	< .001
Theta-Beta Ratio:				
Individual Peak				
Alpha Frequency	-0.34	0.04	9.0	< .001
Age	-0.02	0.003	5.8	< .001
Individual Peak				
Alpha Frequency:				
Theta-Beta Ratio	-0.71	0.08	9.0	< .001
Age	0.03	0.005	5.4	< .001

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